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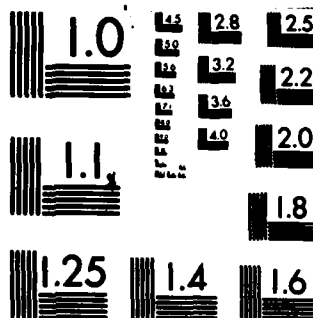
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during the saline experiments. Pre-acclimation, \dot{M}_{sw} was reduced ($p < 0.01$) 65% ($151 \text{ g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$) with atropine, which resulted in higher ($p < 0.01$) T_{re} (0.4°C) and T_{sk} (2.8°C). HR was increased 48% ($53 \text{ b} \cdot \text{min}^{-1}$) by atropine pre-acclimation ($p < 0.01$). Post-acclimation, atropine reduced ($p < 0.01$) \dot{M}_{sw} 33% ($100 \text{ g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$) and increased ($p < 0.01$) HR 63% ($62 \text{ b} \cdot \text{min}^{-1}$) compared to saline exposures. The change in T_{re} per minute ($\Delta T_{re}/\Delta t$) was lower ($p < 0.05$) in atropine-injected subjects following heat acclimation, and their work time was improved by an average of 23.5 minutes ($p = 0.08$). These data demonstrate that heat acclimation improves the endurance time of atropine-treated subjects in a hot-dry environment. This improvement was in part due to the potentiation of sweat gland activity enabling greater evaporative cooling for the same dose of atropine.

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EFFECTS OF HEAT ACCLIMATION ON ATROPINE IMPAIRED THERMOREGULATION

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Running head: Atropine and heat acclimation

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ABSTRACT

The effects of saline or atropine injection (2 mg, im) on eccrine sweating and performance time in seven healthy male subjects were evaluated during treadmill walking ($1.34 \text{ m} \cdot \text{s}^{-1}$) in a hot-dry environment ($T_a = 49^\circ\text{C}$, $T_{dp} = 20.5^\circ\text{C}$) before and after heat acclimation (HA). Mean skin temperature (\bar{T}_{sk}), rectal temperature (T_{re}), and heart rate (HR) were continuously measured. Sweat loss from the skin (\dot{M}_{sw}) was calculated by changes in body weight. HA resulted in decreased ($p < 0.05$) T_{re} (0.4°C) and HR ($17 \text{ b} \cdot \text{min}^{-1}$), and increased ($p < 0.05$) \dot{M}_{sw} ($16 \text{ g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$) during the saline experiments. Pre-acclimation, \dot{M}_{sw} was reduced ($p < 0.01$) 65% ($151 \text{ g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$) with atropine, which resulted in higher ($p < 0.01$) T_{re} (0.4°C) and \bar{T}_{sk} (2.8°C). HR was increased 48% ($53 \text{ b} \cdot \text{min}^{-1}$) by atropine pre-acclimation ($p < 0.01$). Post-acclimation, atropine reduced ($p < 0.01$) \dot{M}_{sw} 33% ($100 \text{ g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$) and increased ($p < 0.01$) HR 63% ($62 \text{ b} \cdot \text{min}^{-1}$) compared to saline exposures. The change in T_{re} per minute ($\Delta T_{re}/\Delta t$) was lower ($p < 0.05$) in atropine-injected subjects following heat acclimation, and their work time was improved by an average of 23.5 minutes ($p = 0.08$). These data demonstrate that heat acclimation improves the endurance time of atropine-treated subjects in a hot-dry environment. This improvement was in part due to the potentiation of sweat gland activity enabling greater evaporative cooling for the same dose of atropine.

Index terms: antimuscarinic drugs, exercise; heat stress; physiological responses

The evaporation of eccrine sweat gland secretion provides the major defense against heat storage in a hot-dry environment (8,17). Eccrine sweat glands are innervated by sympathetic cholinergic fibers, and the primary neurotransmitter substance is acetylcholine (4,16). The responsiveness of the eccrine sweat glands to a given amount of acetylcholine is enhanced after heat acclimation (4,8), which may be the reason for the potentiation of sweat gland activity seen after acclimation to dry heat (7).

Atropine sulfate is useful in the treatment of poisoning by anticholinesterase agents such as organophosphates. Atropine's action is to counteract the effects of accumulation of acetylcholine (15). Its action results from competitive inhibition of acetylcholine at the receptor sites, resulting in a transient blockage of the efferent signal from the central nervous system. In the case of eccrine sweat glands, the result of this competitive inhibition is reduced thermoregulatory sweating causing increased heat storage which may lead to hyperpyrexia (5,13). This effect could be particularly prevalent in soldiers who may have to undergo vigorous exercise in a combat situation following atropine administration.

This study reports on the effects of heat acclimation during exercise-heat exposure following atropine administration. Furthermore, some advantages conferred by heat acclimation, including increased total body sweat rate and prolonged work time in atropine-treated individuals, are discussed.

METHODS

Subjects

Seven healthy male volunteers participated in this investigation after giving their informed consent. The subjects had a mean (\pm SD) age of 22 ± 3 years, weight of 79.9 ± 9.8 kg, Dubois body surface area of $2.02 \pm .16$ m², and

percent body fat (hydrostatic weighing method) of $14.6 \pm 4.6\%$. Their mean maximal aerobic power was $50.2 \pm 6.0 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$.

Experimental Design

The subjects were tested on four separate occasions: two times prior to a heat acclimation program and two times after the completion of that program. The testing consisted of an exercise-heat exposure ($T_a = 49^\circ\text{C}$, $T_{dp} = 20.5^\circ\text{C}$) in which the subjects walked ($1.34 \text{ m} \cdot \text{s}^{-1}$) on a level motor driven treadmill for a prolonged period (4 repeats of 10 minutes of rest, 25 minutes of exercise) or until rectal temperature (T_{re}) exceeded 39.5°C , heart rate (HR) exceeded $180 \text{ b} \cdot \text{min}^{-1}$, or the subject voluntarily terminated the exercise-heat exposure. During the exposures, T_{re} and mean skin (3) temperature (\bar{T}_{sk} ; chest, arm, calf), HR, and assessment (10) of thermal stress (TS) were monitored. Metabolic heat production (M) was calculated during each 25-minute period from open circuit spirometry measurements. Subjects drank water ad libitum during all heat exposures. Total body sweating rates (\dot{M}_{sw}) were determined from body weight changes, corrected for water ingested, measured on a Sauter balance each 25-minute period. \dot{M}_{sw} was corrected for evaporative and convective loss from the respiratory tract (9).

Each subject completed two exercise-heat exposures during the pre-acclimation period. On one occasion, 2 mg of atropine sulfate was injected into the vastus lateralis immediately before the subjects entered the environmental chamber (15 minutes before the onset of exercise). On the other pre-acclimation exposure, subjects were injected with an identical volume (1 ml) of sterile saline. These exposures were randomized and separated by two days to avoid effects of consecutive days of heat exposure.

After the pre-acclimation tests, the subjects completed a 10-day heat acclimation program ($T_a = 49^\circ\text{C}$, $T_{dp} = 20.5^\circ\text{C}$). During this program they

walked on a level treadmill at $1.34 \text{ m} \cdot \text{s}^{-1}$ for 2 repeats of 10 minutes of rest followed by 50 minutes of exercise (120 minutes total) or until voluntary termination. Heat acclimation was defined by equivalent rectal temperatures and heart rates on two consecutive acclimation days. Subjects drank water ad libitum during these exercise exposures.

The post-acclimation testing was identical to the pre-acclimation testing. The subjects were tested on two occasions, once after atropine (2 mg) administration and again after the injection of an equal volume of saline. These exposures were randomized and separated by one day to maintain the level of heat acclimation.

The rate of heat storage (ΔQ) during the exercise-heat exposure was calculated as:

$$\Delta Q = (\Delta T_{re} / \Delta t) (60 \times 0.97 m_b) / A_D \quad (\text{W} \cdot \text{m}^{-2})$$

where:

$\Delta T_{re} / \Delta t$ is the change in rectal temperature per minute

0.97 is the specific heat content of the tissues ($\text{W} \cdot \text{h}^{-1} / \text{kg}^{-1} \cdot ^\circ\text{C}^{-1}$)

m_b is the lean body mass (kg)

A_D is the Dubois surface area (m^2)

Statistical Treatment

Work times for the pre- and post-acclimation atropine experiments were compared by paired t-tests. A 3-way analysis of variance (20) was utilized for all data at the time of peak drug effects (30 minutes post-injection) (15) when a complete heat balance could be determined (14). Linear regression coefficients were calculated for changes in heat storage and rectal temperature over time. An analysis of variance of the slopes of the regression lines, T_{re} vs time and ΔQ vs time, for the four treatments was performed (1). Post-hoc comparisons were accomplished by the Tukey-Kramer method (20). Stepwise regressions of rectal

temperature, performance time and thermal sensation were evaluated (20). Data in the text, tables, and figures are presented as means \pm SD.

RESULTS

All seven subjects were able to complete the control (saline) exercise-heat exposure both pre- and post-heat acclimation. Heat acclimation resulted in significant decreases in final T_{re} (0.4°C , $p < 0.05$), and final HR ($17 \text{ b} \cdot \text{min}^{-1}$, $p < 0.05$), and significantly increased \dot{M}_{sw} ($16 \text{ g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$, $p < 0.05$). Post acclimation, subjects drank more water ($p < 0.05$) during both the saline ($\Delta 383 \text{ ml}$) and atropine ($\Delta 176 \text{ ml}$) exercise-heat exposures.

Exposure time increased by an average of 23.5 minutes (56.5 to 80.0 minutes; $p = 0.08$) during atropine tests post-acclimation. Pre-acclimation, after atropine injection, exercise was terminated by high T_{re} ($> 39.5^{\circ}\text{C}$) in three subjects, high HR ($> 180 \text{ b} \cdot \text{min}^{-1}$) in two subjects, and syncope in the remaining two subjects. After acclimation, exposure time was limited by elevated T_{re} in two subjects, high HR in three subjects, while two subjects completed the 140 minutes of exercise-heat exposure after atropine injection.

Table 1 illustrates the mean data (\pm SD) for the subjects at the 30th minute of exercise-heat exposure pre- and post-acclimation. This point in time was chosen, due to differences in individual subject termination, as it is the last point in the experiment that all data necessary for a complete thermal evaluation of the seven subjects was obtained under all four test conditions. Additionally, the peak effect of atropine on sweat gland inhibition occurred at 30 minutes of exercise. The effects of atropine injection are clearly evident by the decreased \dot{M}_{sw} and increased T_{re} and T_{sk} compared to saline injections. Atropine reduced ($p < 0.05$) pre-acclimation \dot{M}_{sw} by $151 \text{ g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$; however, after heat acclimation atropine reduced \dot{M}_{sw} by only $100 \text{ g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$.

Heat acclimation increased \dot{M}_{sw} ($p < 0.05$) during both control ($27 \text{ g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$) and atropine ($78 \text{ g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$) exercise-heat exposures (Table 2). This table clearly demonstrates that heat acclimation elevated \dot{M}_{sw} for the majority of subjects during both the atropine and saline tests. There was no interaction between the acclimation effect and the drug effects for any of the examined variables.

INSERT TABLES 1 AND 2 ABOUT HERE

The time course of the heart rate responses for the atropine exposures is shown in Figure 1, and rectal temperature responses in Figure 2. Regression analyses of portions of the individual rectal temperature lines during peak drug effects (minutes 24-34) illustrate the change in rectal temperature ($\Delta T_{re}/\Delta t$) was significantly higher ($p < 0.05$) after atropine than saline administration both before and after heat-acclimation. Analysis of variance of the rate of rise in core temperature, pre- and post-acclimation in atropine injected subjects, indicated a significantly lower ($p < 0.05$) core temperature response following acclimation. As would be predicted from the increased rate of change in rectal temperature, the rate of heat storage (Table 3) was significantly greater ($p < 0.05$) in the atropine-treated individuals both pre- and post-acclimation.

INSERT FIGURES 1 AND 2 AND TABLE 3 ABOUT HERE

The change in T_{re} responses pre- to post-acclimation resulting from enhanced \dot{M}_{sw} in atropine injected subjects accounts for 88% of the variance observed in the difference in performance time pre- to post-acclimation based on multiple regression analysis. The addition of other variables (TS, etc.) did not explain any more of the variance in the improved performance time.

DISCUSSION

This investigation provides evidence that heat acclimation can increase the length of time men can exercise at a low work intensity in a hot-dry environment ($T_a = 49^{\circ}\text{C}$, $T_{dp} = 20.5^{\circ}\text{C}$) after atropine injection. The dry heat stress evaluated in this study necessitated evaporative heat loss as the primary heat exchange mechanism (2,8,17). The injection of atropine depressed sweating, resulting in increased heat storage which effectively reduced the time that our subjects could remain in the exercise-heat condition.

The probable mechanism for the increased endurance time in heat-acclimated subjects treated with atropine is potentiation of the sweat glands. The greater efferent drive to the sweat gland in heat-acclimated subjects resulted in an increased amount of acetylcholine which competed with atropine at the effector junction (2,8) and an increase in the cholinergic sensitivity of the eccrine sweat gland (19). Therefore, thermoregulatory sweating can occur more readily following heat acclimation in subjects treated with atropine, enabling longer exercise-heat exposures. The increased sweating rates and lower skin temperatures in atropine-treated subjects following heat acclimation in this study are consistent with this hypothesis (Table 1). Craig *et al.* (5) have previously examined the effect of heat acclimation on atropine-induced inhibition of sweating for resting subjects at 41 and 52°C. Their data indicated that passive heat acclimation did not alter the sweating response under the influence of atropine (2 mg) at rest, suggesting that the mode of acclimation in their study did not result in the sweat gland potentiation as seen in the present study.

The responses of our subjects concurred with those of Metcalf (15) concerning the time course in plasma atropine concentration in that the peak effects of the drug on sweat secretion and heart rate occurred approximately

thirty minutes following the atropine administration. The maximum effect of atropine on heart rate (Figure 1) and sweating rate (Tables 1,2) were similar to that reported in other investigations (6,18). These responses were maximal after approximately thirty minutes of heat exposure. Similarly, the time course of the increase in rectal temperature (Figure 2) was similar to previous studies (5,18), in which heat and/or exercise were evaluated. Higher doses of atropine (3-5 mg, im) do not change the time course of the drug's effect on heart rate, but do increase the magnitude, but not the time, of the rise in core temperature during exercise in a warm environment (6,18). Therefore comparisons of physiological measurements at thirty minutes post-injection (Table 1) enabled evaluation of peak drug effects for all four treatments.

Increased heat storage in atropine-treated subjects during work in a hot environment could potentially result in greater performance decrements and increased heat casualties. Consequently, physical training, heat acclimation, or special equipment that would ameliorate the effect of atropine could be useful. The present data suggests that prior heat acclimation reduces the rate of heat storage during exercise exposure to hot-dry environmental conditions and increases performance time. This decreased heat storage comes from enhanced sweating resulting in better heat dissipation in acclimated individuals treated with atropine. These data have application in the interpretation of previous studies concerning the effects of atropine on exercise-heat stress which require consideration of state of acclimation.

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The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as an official Department of the Army position, policy, or decision, unless so distinguished by other official documentation.

Human subjects participated in these studies after giving their informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

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Table 1. Mean (\pm SD) Values for Seven Male Subjects at the 30th Minute of Heat Exposure

Treatment	T_{re} ($^{\circ}\text{C}$)	\bar{T}_{sk} ($^{\circ}\text{C}$)	M ($\text{W} \cdot \text{m}^{-2}$)	\dot{M}_{sw} ($\text{g} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$)	HR ($\text{b} \cdot \text{min}^{-1}$)
Pre-acclimation					
Saline	37.4 (.2)	35.9 (.9)	185 (24)	280 (54)	110 (9)
Atropine	37.8** (.6)	38.7** (.7)	195 (27)	129** (23)	163** (13)
Post-acclimation					
Saline	37.2 (.2)	36.0 (.8)	174 (19)	307** (39)	98 (13)
Atropine	37.7 (.2)	38.0*** (.7)	185 (22)	207*** (63)	160 (16)

Significantly different from pre-acclimation saline ($p < 0.01$)*Significantly different from pre-acclimation atropine ($p < 0.01$)

Table 2 Individual Total Body Sweat Loss (\dot{M}_{sw} ; $g \cdot m^{-2} \cdot h^{-1}$) at 30 Minutes of Exercise-Heat Exposure, Pre- and Post-acclimation

Subject	Saline		Atropine	
	Pre	Post	Pre	Post
1	262	278	201	177
2	240	271	73	80
3	375	367	103	240
4	235	257	114	157
5	331	328	248	250
6	240	329	130	269
7	274	314	37	278

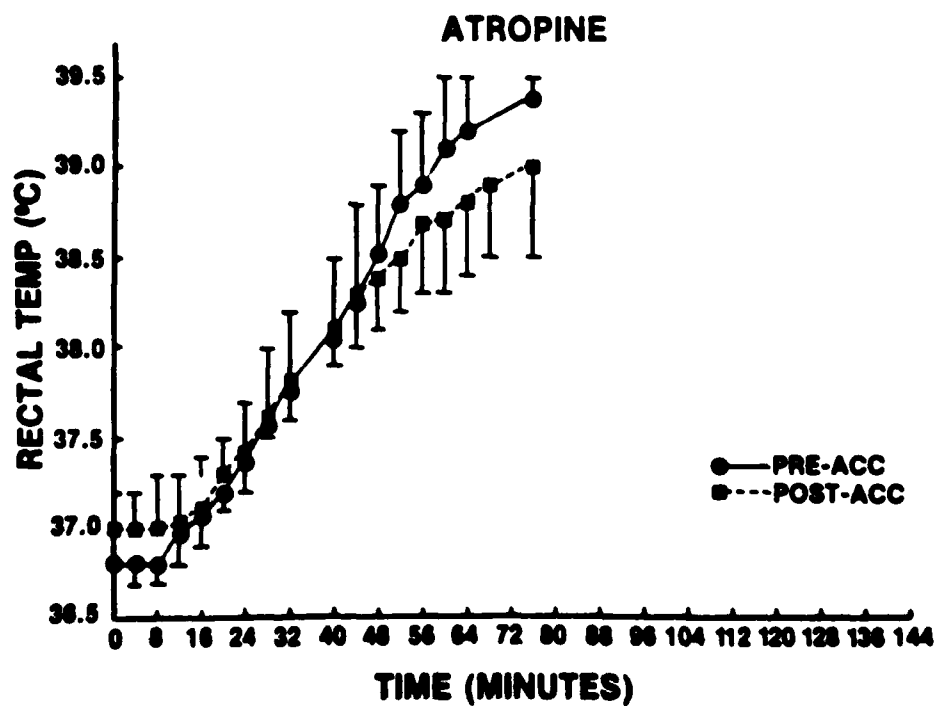
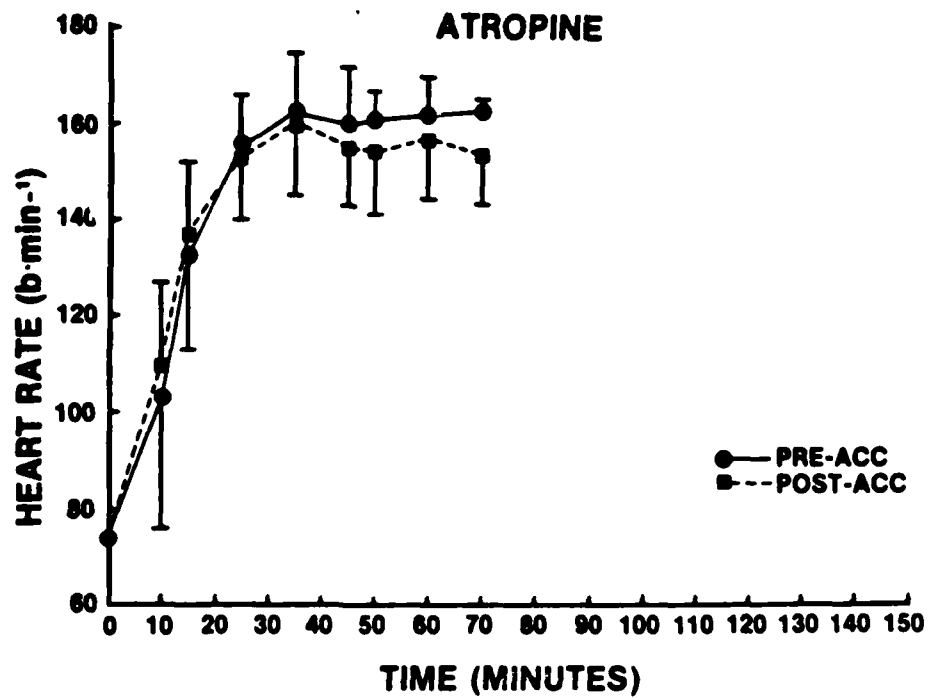
Table 3. The Mean (\pm SD) Rate of Heat Storage ($W \cdot h^{-1}/kg^{-1} \cdot ^\circ C^{-1}$) at Minutes 14 to 24 of Exercise Pre-and Post-acclimation

<u>Pre-acclimation</u>		<u>Post-acclimation</u>	
Saline	Atropine	Saline	Atropine
39.8 \pm 10.1	100.9 \pm 18.7*	36.0 \pm 12.3	92.8 \pm 21.3*

*Significantly different ($p < 0.01$) from saline treatment

FIGURE LEGENDS

- Figure 1 Mean \pm SD heart rate after injection of 2 mg atropine sulfate (i.m.) both pre- and post-heat acclimation.
- Figure 2 Mean \pm SD rectal temperature in 7 males after 2 mg atropine sulfate (i.m.) pre- and post-heat acclimation.



HUMAN RESEARCH

Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

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